09821,160

Freeform Search

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Term: L1 and translat$3 and transcri$7

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DATE: Friday, February 13, 2004 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L2	L1 and translat$3 and transcri$7	6	L2
L1	identif$7 near5 (polypeptide$1 or protein$1) near5 (contact$3 or bind$3) near5 (on or within) near5 cell$1	11	L1

END OF SEARCH HISTORY

09/821,160

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 * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 14:27:38 ON 13 FEB 2004

=> file caplus biosis medline biosis
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                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

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FILE 'BIOSIS' ENTERED AT 14:27:57 ON 13 FEB 2004
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FILE 'MEDLINE' ENTERED AT 14:27:57 ON 13 FEB 2004

=> s translat###(10a)identif########(10a)(polypeptide or protein#)(10a)(on or
within)(10a)cell#
L1            0 TRANSLAT###(10A) IDENTIF########(10A)(POLYPEPTIDE OR PROTEIN#)(1
                0A)(ON OR WITHIN)(10A) CELL#

=> s identif#######(10a)protein#(10A)(on or within)(10a)cell#
L2          419 IDENTIF#######(10A) PROTEIN#(10A)(ON OR WITHIN)(10A) CELL#

=> s l2 and (translat###(10a)trancri######)
L3            0 L2 AND (TRANSLAT###(10A) TRANCRI######)

=> s l2 and (translat####(10a)transcri#####)
L4            0 L2 AND (TRANSLAT####(10A) TRANSCRI#####)

=> s l2 and polymerase chain reaction#
L5           17 L2 AND POLYMERASE CHAIN REACTION#

=> s l5 and translat### and trancri#######
L6            0 L5 AND TRANSLAT### AND TRANCRI#######

=> s l2 and translat### and transcri#######
L7           10 L2 AND TRANSLAT### AND TRANSCRI#######

=> s l7 and polymerase chain reaction#
L8            1 L7 AND POLYMERASE CHAIN REACTION#

=> d l8 bib ab kwic

L8   ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN   2001:151519  BIOSIS
DN   PREV200100151519
TI   Gene function analysis by amber stop codon suppression: CMBF is a nuclear
     protein that supports growth and development of Dictyostelium amoebae.
AU   Winckler, Thomas [Reprint author]; Trautwein, Christine; Tschepke,
     Christina; Neuhaeuser, Christin; Zuendorf, Ilse; Beck, Peter; Vogel,
     Guenter; Dingermann, Theodor
CS   Biozentrum, Institut fuer Pharmazeutische Biologie, Universitaet
     Frankfurt/M., D-60439, Frankfurt am Main, Germany
     winckler@em.uni-frankfurt.de
SO   Journal of Molecular Biology, (26 January, 2001) Vol. 305, No. 4, pp.
     703-714. print.
     CODEN: JMOBAK. ISSN: 0022-2836.
DT   Article
LA   English
ED   Entered STN: 28 Mar 2001
```

Last Updated on STN: 15 Feb 2002

AB The C-module-binding factor, CMBF, is a nuclear DNA-binding **protein** which was originally **identified** through its specific binding to a promoter element **within** the retrotransposable element TRE5-A of Dictyostelium discoideum AX2 **cells**. In order to analyse putative physiological functions of CMBF for the TRE5-A-hosting D. discoideum cells, we used a novel strategy to create mutant cell lines which stably underexpressed functional CMBF. An amber (UAG) **translation** stop codon was introduced into the chromosomal copy of the CMBF-encoding gene (cbfA), and an amber suppressor tRNA gene was expressed in the same mutant cells. Due to the low efficiency of **translation** stop codon suppression in this system all recovered cell lines expressed <20% of wild-type CMBF levels. The mutant cell lines displayed strong growth phenotypes when plated on their natural food source, bacteria. We show evidence that growth reduction was due to impaired phagocytosis of bacteria in the mutants. All obtained mutants showed a strong developmental defect which was defined by the formation of very small fruiting bodies. The strength of the developmental phenotype appeared to depend upon the residual CMBF levels maintained in the mutants. We propose that CMBF is a general **transcription** regulator which supports the normal expression of several genes required for the maintenance of high proliferation rates of D. discoideum amoebae as well as proper aggregation and development. Our results demonstrate that amber stop codon suppression may be a useful strategy to stably underexpress proteins whose coding genes cannot be successfully disrupted by homologous recombination.

AB The C-module-binding factor, CMBF, is a nuclear DNA-binding **protein** which was originally **identified** through its specific binding to a promoter element **within** the retrotransposable element TRE5-A of Dictyostelium discoideum AX2 **cells**. In order to analyse putative physiological functions of CMBF for the TRE5-A-hosting D. discoideum cells, we used a novel strategy to create mutant cell lines which stably underexpressed functional CMBF. An amber (UAG) **translation** stop codon was introduced into the chromosomal copy of the CMBF-encoding gene (cbfA), and an amber suppressor tRNA gene was expressed in the same mutant cells. Due to the low efficiency of **translation** stop codon suppression in this system all recovered cell lines expressed <20% of wild-type CMBF levels. The mutant cell lines. . . phenotype appeared to depend upon the residual CMBF levels maintained in the mutants. We propose that CMBF is a general **transcription** regulator which supports the normal expression of several genes required for the maintenance of high proliferation rates of D. discoideum. . .

IT Methods & Equipment
PCR [**polymerase chain reaction**]: DNA amplification, amplification method, in-situ recombinant gene expression detection, sequencing techniques; electrophoretic mobility shift assay: gene mapping method, restriction fragment. . .

```
=> s l2 and PCR
L9          12 L2 AND PCR

=> s l9 and translat### and transcript###
L10          1 L9 AND TRANSLAT### AND TRANSCRIPT###

=> d l10 bib ab

L10  ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN   2001:151519  BIOSIS
DN   PREV200100151519
TI   Gene function analysis by amber stop codon suppression: CMBF is a nuclear
     protein that supports growth and development of Dictyostelium amoebae.
AU   Winckler, Thomas [Reprint author]; Trautwein, Christine; Tschepke,
```

Christina; Neuhaeuser, Christin; Zuendorf, Ilse; Beck, Peter; Vogel, Guenter; Dingermann, Theodor
CS Biozentrum, Institut fuer Pharmazeutische Biologie, Universitaet Frankfurt/M., D-60439, Frankfurt am Main, Germany
winckler@em.uni-frankfurt.de
SO Journal of Molecular Biology, (26 January, 2001) Vol. 305, No. 4, pp. 703-714. print.
CODEN: JMOBAK. ISSN: 0022-2836.
DT Article
LA English
ED Entered STN: 28 Mar 2001
Last Updated on STN: 15 Feb 2002
AB The C-module-binding factor, CMBF, is a nuclear DNA-binding **protein** which was originally **identified** through its specific binding to a promoter element **within** the retrotransposable element TRE5-A of Dictyostelium discoideum AX2 **cells**. In order to analyse putative physiological functions of CMBF for the TRE5-A-hosting D. discoideum cells, we used a novel strategy to create mutant cell lines which stably underexpressed functional CMBF. An amber (UAG) **translation** stop codon was introduced into the chromosomal copy of the CMBF-encoding gene (cbfA), and an amber suppressor tRNA gene was expressed in the same mutant cells. Due to the low efficiency of **translation** stop codon suppression in this system all recovered cell lines expressed <20% of wild-type CMBF levels. The mutant cell lines displayed strong growth phenotypes when plated on their natural food source, bacteria. We show evidence that growth reduction was due to impaired phagocytosis of bacteria in the mutants. All obtained mutants showed a strong developmental defect which was defined by the formation of very small fruiting bodies. The strength of the developmental phenotype appeared to depend upon the residual CMBF levels maintained in the mutants. We propose that CMBF is a general **transcription** regulator which supports the normal expression of several genes required for the maintenance of high proliferation rates of D. discoideum amoebae as well as proper aggregation and development. Our results demonstrate that amber stop codon suppression may be a useful strategy to stably underexpress proteins whose coding genes cannot be successfully disrupted by homologous recombination.

09/821,160

```
=> s (identif####### or detect###)(10a)(polypeptide# or protein#)(10a)(contact###
cell####### membrane# or on cell# or within cell#)
   1 FILES SEARCHED...
   2 FILES SEARCHED...
L1        62424 (IDENTIF####### OR DETECT###)(10A)(POLYPEPTIDE# OR PROTEIN#)(10A
                )(CONTACT### CELL####### MEMBRANE# OR ON CELL# OR WITHIN CELL#)

=> s l1 and (translat###(10a)transcri#######)
L2          586 L1 AND (TRANSLAT###(10A) TRANSCRI#######)

=> s (identif####### or detect###)(5A)(polypeptide# or protein#)(5a)(contact### or
bind###)(5a)(cell####### membrane or on cell# or within cell#)
   1 FILES SEARCHED...
   2 FILES SEARCHED...
L3         2422 (IDENTIF####### OR DETECT###)(5A)(POLYPEPTIDE# OR PROTEIN#)(5A)(
                CONTACT### OR BIND###)(5A)(CELL####### MEMBRANE OR ON CELL# OR
                WITHIN CELL#)

=> s l3 and (translat###(5A)transcri#######)
L4           18 L3 AND (TRANSLAT###(5A) TRANSCRI#######)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5             8 DUP REM L4 (10 DUPLICATES REMOVED)

=> d l5 1-8 bib ab kwic

L5   ANSWER 1 OF 8     MEDLINE on STN                    DUPLICATE 1
AN   2002147235     MEDLINE
DN   21874805   PubMed ID: 11878877
TI   Analysis of early region 1 of porcine adenovirus type 3.
AU   Zhou Y; Tikoo S K
CS   Virology Group, University of Saskatchewan, Saskatoon, Saskatchewan, S7N
     5E3, Canada.
SO   VIROLOGY, (2001 Dec 5) 291 (1) 68-76.
     Journal code: 0110674. ISSN: 0042-6822.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200204
ED   Entered STN: 20020308
     Last Updated on STN: 20020403
     Entered Medline: 20020401
AB   To identify the proteins encoded by the porcine adenovirus 3 (PAV-3) E1
     region, rabbit antisera were prepared using a bacterial fusion protein
     encoding E1A, E1B(small), or E1B(large) protein. Sera against E1A,
     E1B(small), and E1B(large) immunoprecipitated a protein of 35, 23, and 53
     kDa, respectively, in in vitro translated and
     transcribed mRNA and PAV-3 infected cells. To determine the role
     of E1 proteins in PAV-3 replication, we constructed vectors with a
     deletion(s) in the E1 region. Mutant PAV211, containing deletions in E1A
     and E3, grew to titers similar to wild-type in VIDO R1 cells (E1A
     complementing) but not in swine testicular (ST) cells. No early protein
     (E1B(small), DNA binding protein) expression could be
     detected in PAV211 infected ST cells by Western blots.
     Mutant PAV212, containing deletions in E1B(small) and E3, grew to
     wild-type titers in VIDO R1 or ST cells. These deletions were
     successfully rescued, resulting in recombinant PAV214, containing
     deletions in E1A, E1B(small), and E3. However, mutant PAV-3, containing a
     triple stop codon inserted in the E1B(large) coding sequence, could not be
     isolated. Next, we constructed a recombinant PAV216 by inserting the
     green fluorescent protein gene flanked by a promoter and a poly(A) in the
     E1A region of the PAV214 genome. Both PAV214 and PAV216 replicate as
```

efficiently as wild-type in VIDO R1 cells. These results suggested that (a) E1A is essential for virus replication and is required for the activation of other PAV-3 early genes, (b) E1B(small) is not essential for replication of PAV-3, and (c) E1B(large) is essential for virus replication. Moreover, the PAV216 vector can be used for the expression of a transgene.

AB . . . protein. Sera against E1A, E1B(small), and E1B(large) immunoprecipitated a protein of 35, 23, and 53 kDa, respectively, in in vitro **translated** and **transcribed** mRNA and PAV-3 infected cells. To determine the role of E1 proteins in PAV-3 replication, we constructed vectors with a. . . to wild-type in VIDO R1 cells (E1A complementing) but not in swine testicular (ST) cells. No early protein (E1B(small), DNA **binding protein**) expression could be **detected** in PAV211 infected ST **cells** by Western blots. Mutant PAV212, containing deletions in E1B(small) and E3, grew to wild-type titers in VIDO R1 or ST. . .

L5 ANSWER 2 OF 8 MEDLINE on STN DUPLICATE 2
AN 96336514 MEDLINE
DN 96336514 PubMed ID: 8738726
TI Primary cultured normal human hepatocytes for hepatitis B virus receptor studies.
AU Mabit H; Vons C; Dubanchet S; Capel F; Franco D; Petit M A
CS Institut National de la Sante de la Recherche Medicale Unite, Unite, Clamart, France.
SO JOURNAL OF HEPATOLOGY, (1996 Apr) 24 (4) 403-12.
Journal code: 8503886. ISSN: 0168-8278.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199612
ED Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961211
AB BACKGROUND/AIMS: We analyzed the hepatitis B virus envelope specificities (HBs, preS2 and preS1) involved in virus attachment to normal human hepatocytes, and we performed in vitro hepatitis B virus infection experiments without addition of dimethyl sulfoxide and polyethylene glycol, which may affect cell membrane integrity, in order to study further the early steps of the life cycle of the hepatitis B virus. METHODS: Primary normal human hepatocytes were prepared from surgical biopsies by the two-step collagenase perfusion technique, and cultured in a fetal calf serum-free medium supplemented with 10(-6) M dexamethasone. Cell-binding assays, ligand blotting and immunohistochemistry experiments were carried out using our anti-idiotypic (Ab2) antibodies (Ab2s/preS1, Ab2s/preS2 and Ab2s/HBs). RESULTS: Probing primary normal human hepatocytes, the 35-kDa major preS1-**binding protein** (preS1-BP35) we have previously **identified** in human hepatoma HepG2 **cells** was recognized in blotting, whereas both HBs- and preS1-specificities of the hepatitis B virus envelope interacted strongly with normal human hepatocyte cell membrane in cell-binding assays and immunohistochemistry experiments. Hepatitis B virus infectivity studies confirmed a great inter-experimental variability depending on donors and liver perfusion, and demonstrated a great intra-experimental variability depending on the serum-derived hepatitis B virus isolate used for the inoculation. In our culture conditions, only increased detection of the RC and CCC DNA forms of hepatitis B virus in cells and of hepatitis B virus surface antigens in medium was observed 4 to 8 days after exposure of cells to hepatitis B virus. CONCLUSION: These findings support a potential role for preS1-BP35 as a receptor protein for hepatitis B virus. In our hands, limitation(s) in the hepatitis B virus life cycle may occur at some step after virion binding, and likely result from complex

regulation of reverse **transcription** of the RNA and **translation** of core protein by extrahepatic host factors or/and by the virus itself. However, the normal human hepatocyte model developed here is available for studying the initial steps in hepatitis B virus entry into cells.

AB . . . carried out using our anti-idiotypic (Ab2) antibodies (Ab2s/preS1, Ab2s/preS2 and Ab2s/HBs). RESULTS: Probing primary normal human hepatocytes, the 35-kDa major preS1-**binding protein** (preS1-BP35) we have previously **identified** in human hepatoma HepG2 **cells** was recognized in blotting, whereas both HBs- and preS1-specificities of the hepatitis B virus envelope interacted strongly with normal human. . . B virus life cycle may occur at some step after virion binding, and likely result from complex regulation of reverse **transcription** of the RNA and **translation** of core protein by extrahepatic host factors or/and by the virus itself. However, the normal human hepatocyte model developed here. . .

L5 ANSWER 3 OF 8 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AN 97183353 EMBASE

DN 1997183353

TI Multiple myosin isozymes and hair-cell function.

AU Gillespie P.G.; Hasson T.; Garcia J.A.; Corey D.P.

CS P.G. Gillespie, Department of Physiology, Johns Hopkins University, Baltimore, MD 21205, United States

SO Cold Spring Harbor Symposia on Quantitative Biology, (1996) 61/- (309-318).
Refs: 79
ISSN: 0091-7451 CODEN: CSHSAZ

CY United States

DT Journal; Conference Article

FS 029 Clinical Biochemistry

LA English

SL English

AB The above enumeration does not conclusively summarize identities and functions of all hair-cell myosin isozymes. The RT-PCR screen described by Sole et al. (1994) demonstrated that other myosin isozymes, including myosin Iα and myosin X, are also found in hair-cell-containing epithelia; in addition, primer sets used may have missed other classes; for example, homologous of myosins III and Vb. Additional cataloging of myosins mRNAs from frog sacculus and other auditory and vestibular tissues will no doubt identify additional myosin isozymes expressed in hair cells. Myosin molecules besides myosin VI and VIIa are likely to be essential for hair cells. Genes for several myosin isozymes, including those for myosins Iβ, Iγ, and VIIb, are predicted to map closely to the chromosomal locations of at least one human deafness gene (Hasson et al. 1996). Mutations in their hair-cell myosin isozymes may not manifest as human deafnesses if the isozyme in question is employed for other essential roles. Although distribution of myosin molecules within the auditory and vestibular systems is complicated, we believe that a specific, principal role can be ascribed to each isozyme. Myosin V is not found in hair cells and likely serves the same role in afferent nerve processes that it plays elsewhere in the nervous system. Myosin I- probably mediates adaptation and may contribute to transport of proteins throughout stereocilia. Myosin VI appears to cross-link actin filaments within the cuticular plate and perhaps between the cuticular plate and stereociliary rootlets; mutations in myosin VI may disrupt these structures and prevent bundle formation. Myosin VIIa may actually hold stereocilia together, mutations in myosin VIIa may therefore cause stereocilia to splay apart, preventing proper assembly into a hair bundle. Myosin Iβ, VI, and VIIa are each at unusually high concentrations in bundles of the most newly formed hair cells, usually at the sensory epithelium's periphery (Gillespie et al. 1993; T. Hasson et al., in

prepare); these three isozymes may therefore play additional, more specialized roles during hair-bundle development (Tilney et al. 1992). In the future, we expect that exciting results will come from experiments designed to test these predictions. A key conclusion from hair- cell myosin localization is that these myosin molecules are not necessarily concentrated in regions of high actin density. Despite the similarity of their catalytic domains, each of the myosin isozymes found in hair cells has a distinctive subcellular localization. Furthermore, each hair-cell myosin isozyme appears in multiple specific locations. The ability of myosin molecules to bind to and translocate along actin filaments must therefore be heavily regulated; for dictating location, the affinity of myosin molecules for other proteins may be just as important as their affinity for actin. The nonhomologous tail domains of each class must contribute substantially to this differential localization. A critical goal for the field is therefore **identification** of hair-**cell proteins** that **bind** to myosin molecules, because their localization and abundance relative to each myosin isozyme may establish the final destination of a given myosin molecule. Finally, we wonder why myosin molecules would be employed in capacities that appear, at first consideration, to be entirely structural. In particular, our hypothesized roles for myosins VI and VIIa-maintaining cuticular-plate and bundle structural integrity-do not require force-producing molecules. Proteins that simply cross-link actin or connect actin to specific membrane receptors would seem to function adequately. Because the cell expends considerable energy to **transcribe** and **translate** relatively large myosin motor domains instead of smaller actin-binding structures, it seems likely that actin-activated ATPase plays a fundamental role in the behavior of these myosin isozymes. Because myosins VI and VIIa are not simply targeted to distal ends of actin filaments, but are instead found in association with specific actin-filament domains, the role of the motor activity must be relatively subtle. It seems likely that by understanding this paradox, we will understand the precise role of each myosin isozyme in the hair cell.

AB . . . tail domains of each class must contribute substantially to this differential localization. A critical goal for the field is therefore **identification** of hair-**cell proteins** that **bind** to myosin molecules, because their localization and abundance relative to each myosin isozyme may establish the final destination of a. . . actin or connect actin to specific membrane receptors would seem to function adequately. Because the cell expends considerable energy to **transcribe** and **translate** relatively large myosin motor domains instead of smaller actin-binding structures, it seems likely that actin-activated ATPase plays a fundamental role. . .

L5 ANSWER 4 OF 8 MEDLINE on STN DUPLICATE 3
AN 94263234 MEDLINE
DN 94263234 PubMed ID: 8203918
TI Overexpression of iron-responsive element-binding protein and its analytical characterization as the RNA-binding form, devoid of an iron-sulfur cluster.
AU Basilion J P; Kennedy M C; Beinert H; Massinople C M; Klausner R D; Rouault T A
CS National Institutes of Health, National Institute of Child Health and Human Development, Bethesda, Maryland 20892.
NC GM 34812 (NIGMS)
SO ARCHIVES OF BIOCHEMISTRY AND BIOPHYSICS, (1994 Jun) 311 (2) 517-22. Journal code: 0372430. ISSN: 0003-9861.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199407
ED Entered STN: 19940714
Last Updated on STN: 19970203

Entered Medline: 19940701

AB The iron-responsive element-binding protein (IRE-BP) has been defined and **identified** as an RNA-**binding protein** found in iron-deprived eukaryotic **cells**. IRE-BP binds to stem-loop structures, iron-responsive elements (IREs), which are located in the untranslated regions of the mRNAs for several genes including ferritin, and the transferrin receptor. When bound, IRE-BP prevents ferritin **translation** and stabilizes the transferrin receptor **transcript**. When cells are iron replete, an iron-sulfur cluster is ligated to the IRE-BP, the protein loses RNA binding properties, and it acquires aconitase activity. Cytosolic aconitase from liver can be converted into the IRE-BP by oxidative removal of its Fe-S cluster. We describe here overexpression of IRE-BP in baculovirus-infected insect cells which yields IRE-BP devoid of an iron-sulfur cluster. We describe a one-step purification of the IRE-BP and a quantitative analysis of Fe, S2-, S0, protein, and enzyme activity on IRE-BP, as obtained in cell lysates, after purification, and after reconstitution to active aconitase. On the average not more than 3% of the over-expressed purified protein contained an intact Fe-S cluster, and it was demonstrated that that cluster was not lost during purification. Scatchard analysis of RNA-binding data was compatible with a single high-affinity RNA-binding form of the IRE-BP. Active aconitase could be reconstituted from the purified IRE-BP obtained from the expression system by addition of iron, thiol, and sulfide, and the characteristic epr spectrum of the 3Fe form of cytosolic aconitase was obtained after ferricyanide oxidation of the reconstituted material.

AB The iron-responsive element-binding protein (IRE-BP) has been defined and **identified** as an RNA-**binding protein** found in iron-deprived eukaryotic **cells**. IRE-BP binds to stem-loop structures, iron-responsive elements (IREs), which are located in the untranslated regions of the mRNAs for several genes including ferritin, and the transferrin receptor. When bound, IRE-BP prevents ferritin **translation** and stabilizes the transferrin receptor **transcript**. When cells are iron replete, an iron-sulfur cluster is ligated to the IRE-BP, the protein loses RNA binding properties, and. . .

L5 ANSWER 5 OF 8 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

AN 93040298 EMBASE

DN 1993040298

TI An essential yeast gene encoding a TTAGGG repeat-binding protein.

AU Brigati C.; Kurtz S.; Balderes D.; Vidali G.; Shore D.

CS Department of Microbiology, College of Physicians and Surgeons, Columbia University, 701 West 168th Street,New York, NY 10032, United States

SO Molecular and Cellular Biology, (1993) 13/2 (1306-1314). ISSN: 0270-7306 CODEN: MCEBD4

CY United States

DT Journal; Article

FS 022 Human Genetics
029 Clinical Biochemistry

LA English

SL English

AB A yeast gene encoding a DNA-binding protein that recognizes the telomeric repeat sequence TTAGGG found in multicellular eukaryotes was identified by screening a λgt11 expression library with a radiolabeled TTAGGG multimer. This gene, which we refer to as TBF1 (TTAGGG repeat-binding factor 1), encodes a polypeptide with a predicted molecular mass of 63 kDa. The TBF1 protein, produced in vitro by **transcription** and **translation** of the cloned gene, binds to (TTAGGG)(n) probes and to a yeast telomeric junction sequence that contains two copies of the sequence TTAGGG separated by 5 bp. TBF1 appears to be identical to a previously described yeast TTAGGG-repeat binding activity called TBFα. TBF1 produced in vitro yields protein-DNA complexes with

(TTAGGG)(n) probes that have mobilities on native polyacrylamide gels identical to those produced by partially purified TBFα from yeast cells. Furthermore, when extracts are prepared from a strain containing a TBF1 gene with an antigen tag, we find that the antigen copurifies with the predominant (TTAGGG)(n)-binding activity in the extracts. The DNA sequence of TBF1 was determined. The predicted protein sequence suggests that TBF1 may contain a nucleotide-binding domain, but no significant similarities to any other known **proteins** were **identified**, nor was an obvious DNA-**binding** motif apparent. Diploid **cells** heterozygous for a tbf1::URA3 insertion mutation are viable but upon sporulation give rise to tetrads with only two viable spores, both of which are Ura-, indicating that the TBF1 gene is essential for growth. Possible functions of TBF1 (TFBα) are discussed in light of these new results.

AB . . . factor 1), encodes a polypeptide with a predicted molecular mass of 63 kDa. The TBF1 protein, produced in vitro by **transcription** and **translation** of the cloned gene, binds to (TTAGGG)(n) probes and to a yeast telomeric junction sequence that contains two copies of. . . The predicted protein sequence suggests that TBF1 may contain a nucleotide-binding domain, but no significant similarities to any other known **proteins** were **identified**, nor was an obvious DNA-**binding** motif apparent. Diploid **cells** heterozygous for a tbf1::URA3 insertion mutation are viable but upon sporulation give rise to tetrads with only two viable spores,. . .

L5 ANSWER 6 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 4

AN 1989:160558 BIOSIS

DN PREV198987082659; BA87:82659

TI BIOCHEMICAL AND IMMUNOLOGICAL COMPARISONS OF CARBOHYDRATE-BINDING PROTEIN 35 AND AN IGE-BINDING PROTEIN.

AU LAING J G [Reprint author]; ROBERTSON M W; GRITZMACHER C A; WANG J L; LIU F-T

CS DIV OF MOL BIOL, MED BIOL INST, 11077 N TORREY PINES RD, LA JOLLA, CALIF 92037, USA

SO Journal of Biological Chemistry, (1989) Vol. 264, No. 4, pp. 1907-1910. CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 25 Mar 1989
Last Updated on STN: 25 Mar 1989

AB The predicted amino acid sequence of carbohydrate-binding protein 35 (CBP35; Mr .apprx. 35,000), a galactose-specific lectin in many mouse and human cells, has been compared to the predicted sequence of an IgE-**binding protein** (εBP) originally **identified** in rat basophilic leukemia **cells**. The sequences of the two proteins showed that: (a) 85% of the amino acid residues are identical; (b) the polypeptide chains are comprised of two distinct domains; and (c) highly conserved internal repetitive sequences are present. Consistent with these observations, antisera raised against CBP35 or against a synthetic peptide derived from the εBP sequence cross-reacted with both proteins. Moreover, fractionation of extracts of mouse 3T3 fibroblasts over an IgE-Sepharose affinity column showed that CBP35 bound to IgE; this binding was reversed by addition of lactose. Conversely, fractionation of extracts of rat basophilic leukemia cells over an affinity column of Sepharose derivatized with N-(ε-aminocaproyl)-D-galactosamine showed that εBP was a galactose-binding protein. Furthermore, εBP bound to IgE-Sepharose could be eluted by lactose. Finally, **transcription** and **translation** in vitro of the cDNA corresponding to εBP yielded a polypeptide containing carbohydrate-binding activity. All of these results strongly support the conclusion that CBP35 and εBP are mouse and rat homologues, respectively.

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IT . . . Descriptors
RAT BASOPHILIC LEUKEMIA CELL HUMAN MOUSE GALACTOSE-SPECIFIC LECTIN MOLECULAR SEQUENCE DATA AMINO ACID SEQUENCE COMPARISON CONSERVED SEQUENCES COMPLEMENTARY DNA **TRANSCRIPTION TRANSLATION** ANTIBODY CROSS REACTION LACTOSE BINDING REVERSAL GALACTOSE BINDING IMMUNOGLOBULIN E

L5 ANSWER 7 OF 8 MEDLINE on STN
AN 89123243 MEDLINE
DN 89123243 PubMed ID: 2536691
TI Biochemical and immunological comparisons of carbohydrate-binding protein 35 and an IgE-binding protein.
AU Laing J G; Robertson M W; Gritzmacher C A; Wang J L; Liu F T
CS Department of Biochemistry, Michigan State University, East Lansing 48824.
NC AI-19747 (NIAID)
GM-27203 (NIGMS)
GM-38740 (NIGMS)
+
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1989 Feb 5) 264 (4) 1097-10.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198903
ED Entered STN: 19900308
Last Updated on STN: 19970203
Entered Medline: 19890313
AB The predicted amino acid sequence of carbohydrate-binding protein 35 (CBP35; Mr approximately 35,000), a galactose-specific lectin in many mouse and human cells, has been compared to the predicted sequence of an IgE-**binding protein** (epsilon BP) originally **identified** in rat basophilic leukemia **cells**. The sequences of the two proteins showed that: (a) 85% of the amino acid residues are identical; (b) the polypeptide chains are comprised of two distinct domains; and (c) highly conserved internal repetitive sequences are present. Consistent with these observations, antisera raised against CBP35 or against a synthetic peptide derived from the epsilon BP sequence cross-reacted with both proteins. Moreover, fractionation of extracts of mouse 3T3 fibroblasts over an IgE-Sepharose affinity column showed that CBP35 bound to IgE; this binding was reversed by addition of lactose. Conversely, fractionation of extracts of rat basophilic leukemia cells over an affinity column of Sepharose derivatized with N-(epsilon-amino-caproyl)-D-galactosamine showed that epsilon BP was a galactose-binding protein. Furthermore, epsilon BP bound to IgE-Sepharose could be eluted by lactose. Finally, **transcription** and **translation** in vitro of the cDNA corresponding to epsilon BP yielded a polypeptide containing carbohydrate-binding activity. All of these results strongly support the conclusion that CBP35 and epsilon BP are mouse and rat homologues, respectively.
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L5 ANSWER 8 OF 8 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1982:246218 BIOSIS
DN PREV198274018698; BA74:18698
TI VIRAL GENE PRODUCTS IN ADENOVIRUS TYPE 2 TRANSFORMED HAMSTER CELLS.
AU ESCHE H [Reprint author]
CS INST GENTICS, UNIV COLOGNE, COLOGNE, FEDERAL REPUBLIC OF GERMANY
SO Journal of Virology, (1982) Vol. 41, No. 3, pp. 1076-1082.
CODEN: JOVIAM. ISSN: 0022-538X.
DT Article
FS BA
LA ENGLISH
AB Viral gene products expressed in 5 adenovirus type 2 (Ad2)-transformed hamster embryo cell lines (Ad2 HE1-5) were analyzed by cell-free translation of cytoplasmic, viral RNA which was selected by hybridization to cloned restriction endonuclease fragments of Ad2 DNA. Proteins synthesized in vitro were analyzed by electrophoresis in sodium dodecyl sulfate-polyacrylamide gels and compared with those directed by RNA prepared from productively infected cells. The early regions E1 and E4 of Ad2 were found to be expressed in all of 5 Ad2-transformed hamster embryo cell lines. RNA transcribed from early region E2, which codes for the 72,000-MW (72K) DNA-**binding protein** was **detected** in **cell** line HE1 only, and early region E3 was expressed exclusively in cell line HE4. RNA transcribed from the region .apprx. 12-35 map units, coding for immediate early (13.5K, 52/53K) and intermediate early proteins (13.6K, 16K, 17K, 87K), and RNA from late genes, was not found in any of the cell lines. The proteins synthesized in vitro by RNA prepared from cell lines HE1 to HE5 had electrophoretic mobilities similar to those programmed by RNA from productively infected cells.
AB. . . all of 5 Ad2-transformed hamster embryo cell lines. RNA transcribed from early region E2, which codes for the 72,000-MW (72K) DNA-**binding protein** was **detected** in **cell** line HE1 only, and early region E3 was expressed exclusively in cell line HE4. RNA transcribed from the region .apprx.. . .
IT Miscellaneous Descriptors
EMBRYO CELLS RNA **TRANSLATION** IMMEDIATE EARLY PROTEINS **TRANSCRIPTION** RESTRICTION ENDO NUCLEASE

=>

09/821,160

side			result set
DB=USPT,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ			
L13	(detect$ or identif$7) near5 (polypeptide$1 or protein$1) near5 on (cell$5 membrance or on cell$1 or within cell$1)	0	L13
L12	ELISA near5 (on cell or on cell$5 membrane)	0	L12
L11	identif$7 near5 (polypeptide$1 or protein$1) near5 (interact$3 or binid$3 or contact$3) near5 (cell$5 membrane$1 or on cell$1 or within cell$1)	13	L11
L10	l8 and bind$ moiety and interact$3	1	L10
L9	L8 and (bind$3 moiety near5 interact$3 domain$1)	0	L9
L8	L7 and (translat$3 near5 transcrib$7)	91	L8
L7	L6 and (identif$7 protein41 or identif$7 polypeptide$1)	135	L7
L6	(interact$3 or bind$3) near5 (cell$5 membrane or on cell$1 or within cell$1)	4167	L6
L5	l4 and (contact$3 cell$1 or bind$3 to cell$1 or contact$3 membrane$1)	0	L5
L4	l3 and ((on or within) cell$1)	2	L4
L3	L2 and (identif$7 near5 (polypeptide$1 or protein$1) near5 (contact$3 or bind$3) near5 (on or within) near5 cell$1)	2	L3
L2	6265545.pn.	2	L2
L1	6265545.pn	0	L1

END OF SEARCH HISTORY

09/821,160.

```
=> s transcri#####(10a)translat###(10a)polymerase chain reaction#
L1          159 TRANSCRI#####(10A) TRANSLAT###(10A) POLYMERASE CHAIN REACTION#

=> s l1 and (identif#######(10a)(polypeptide# or protein# or peptide#)0
MISSING OPERATOR PEPTIDE#)0
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l1 and (identif#######(10a)(polypeptide# or protein# or peptide#))
L2           13 L1 AND (IDENTIF#######(10A)(POLYPEPTIDE# OR PROTEIN# OR PEPTIDE#
                ))

=>

=> s l2 and bind###
L3            4 L2 AND BIND###

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4             1 DUP REM L3 (3 DUPLICATES REMOVED)

=> d l4 bib ab kwic

L4   ANSWER 1 OF 1     MEDLINE on STN                    DUPLICATE 1
AN   90138861      MEDLINE
DN   90138861    PubMed ID: 2153955
TI   DNA amplification-restricted transcription-translation: rapid analysis of
     rhesus rotavirus neutralization sites.
CM   Erratum in: Proc Natl Acad Sci U S A 1990 Jun;87(11):4411
AU   Mackow E R; Yamanaka M Y; Dang M N; Greenberg H B
CS   Department of Medicine, Stanford University, CA 94305.
NC   R22 AI1362 (NIAID)
SO   PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF
     AMERICA, (1990 Jan) 87 (2) 518-22.
     Journal code: 7505876. ISSN: 0027-8424.   Request a copy 2/12/2004
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   199003
ED   Entered STN: 19900328
     Last Updated on STN: 19970203
     Entered Medline: 19900306
AB   DNA amplification-restricted transcription-translation
     (DARTT), is based on DNA amplification by the polymerase
     chain reaction (PCR) and uses PCR to truncate
     protein-encoding DNA while adding transcriptional and translational
     initiation signals to the segment. The amplified DNA segments are
     transcribed into RNA and translated into protein in vitro and the
     synthesized proteins are used to define functional sites. DARTT was
     applied to rhesus rotavirus gene segment 4 cDNA in order to create a
     series of carboxyl-terminal truncations and new amino termini in the
     encoded VP4 capsid protein. The truncated VP4 polypeptides were tested
     for reaction with 11 VP4-specific neutralizing monoclonal antibodies to
     identify the minimum polypeptides required for antibody
     recognition. Monoclonal antibodies 2G4, M2, and M7, which neutralize a
     number of serologically distinct rotaviruses, required amino acids 247-474
     of VP4 for binding. DARTT is potentially applicable to the
     identification of discontinuous epitopes and functional domains on
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```
=> s yu.in.
L5         5753 YU.IN.

=> s l5 and Zhongping
L6            0 L5 AND ZHONGPING
```